PART 8

# THE HEART

CHAPTER 36

# Inspection of the Neck Veins

## Key Teaching Points

- In patients with dyspnea, ascites, or edema, determination of venous pressure at the bedside is essential. If venous pressure is elevated, the patient has cardiopulmonary disease; if venous pressure is normal, liver or kidney disease is likely.
- Bedside estimates of venous pressure are accurate when compared with measured values.
- In patients with chest pain or dyspnea, elevated neck veins increase the probability of elevated left heart pressure and depressed ejection fraction.
- The most important feature that distinguishes the internal jugular venous waveform from arterial movements is its conspicuous *inward* movement (arterial movements have a conspicuous *outward* movement).
- Kussmaul sign and the positive abdominojugular test often appear together. They are found in patients with constrictive pericarditis and right ventricular infarction and some with severe heart failure. In heart failure, Kussmaul sign is associated with an unfavorable prognosis.

## I. INTRODUCTION

Clinicians should inspect the neck veins for the following reasons: (1) to detect elevated central venous pressure (CVP) and (2) to detect specific abnormalities of venous waveforms, which are characteristic of certain arrhythmias and some valvular, pericardial, and myocardial disorders.

Clinicians first associated conspicuous neck veins with heart disease approximately 3 centuries ago.[1,2] In the late 1800s Sir James Mackenzie described venous waveforms of arrhythmias and various heart disorders, using a mechanical polygraph applied over the patient's neck or liver. His labels for the venous waveforms—A, C, and V waves—are still used today.[3,4] Clinicians began to estimate venous pressure at the bedside routinely in the 1920s, after the introduction of the glass manometer and after Starling's experiments linking venous pressure to cardiac output.[5]

# II. VENOUS PRESSURE

## A. DEFINITIONS

### 1. CENTRAL VENOUS PRESSURE

Central venous pressure (CVP) is mean vena caval or right atrial pressure, which, in the absence of tricuspid stenosis, equals right ventricular end-diastolic pressure. Disorders that increase diastolic pressures of the right side of the heart—left heart disease, lung disease, primary pulmonary hypertension, and pulmonic stenosis—all increase the CVP and make the neck veins abnormally conspicuous. CVP is expressed in millimeters of mercury (mm Hg) or centimeters (cm) of water above atmospheric pressure (1.36 cm water = 1.0 mm Hg).

Estimations of CVP are most helpful in patients with ascites or edema, in whom an elevated CVP indicates heart or lung disease and a normal CVP suggests alternative diagnoses, such as chronic liver disease. Despite the prevailing opinion, the CVP is normal in patients with liver disease; the edema in these patients results from hypoalbuminemia and the weight of ascites compressing veins to the legs.[6-9]

### 2. PHYSIOLOGIC ZERO POINT

Physiologists have long assumed that a location in the cardiovascular system (presumed to be the right atrium in humans) tightly regulates venous pressure so that it remains the same even when the person changes position.[5,10-12] All measurements of CVP—whether by clinicians inspecting neck veins or by catheters in intensive care units—attempt to identify the pressure at this zero point (e.g., if a manometer connected to a systemic vein supports a column of saline 8 cm above the zero point, with the top of the manometer open to atmosphere, the recorded pressure in that vein is 8 cm water). Estimates of CVP are related to the zero point because interpretation of this value does not need to consider the hydrostatic effects of different patient positions, and any abnormal value thus indicates disease.

### 3. EXTERNAL REFERENCE POINT

Clinicians require some external reference point to reliably locate the level of the zero point. Of the many such reference points that have been proposed over the past century,[5] only two are commonly used today: the sternal angle and phlebostatic axis.

#### A. STERNAL ANGLE

In 1930 Sir Thomas Lewis, a pupil of Mackenzie, proposed a simple bedside method for measuring venous pressure designed to replace the manometer, which he found too burdensome for general use.[13] He observed that the top of the jugular veins of normal persons (and the top of the fluid in the manometer) always came to lie within 1 to 2 cm of vertical distance from the sternal angle, whether the person was supine, semiupright, or upright (an observation since confirmed by others).[14] If the top level of the neck veins was more than 3 cm above the sternal angle, Lewis concluded the venous pressure was elevated.

Others have modified this method, stating that the CVP equals the vertical distance between the top of the neck veins and a point 5 cm below the sternal angle (Fig. 36.1).[15] This variation is commonly called the method of Lewis, although Lewis never made such a claim.

#### B. PHLEBOSTATIC AXIS

The phlebostatic axis is the midpoint between the anterior and posterior surfaces of the chest at the level of the fourth intercostal space. This reference point, the

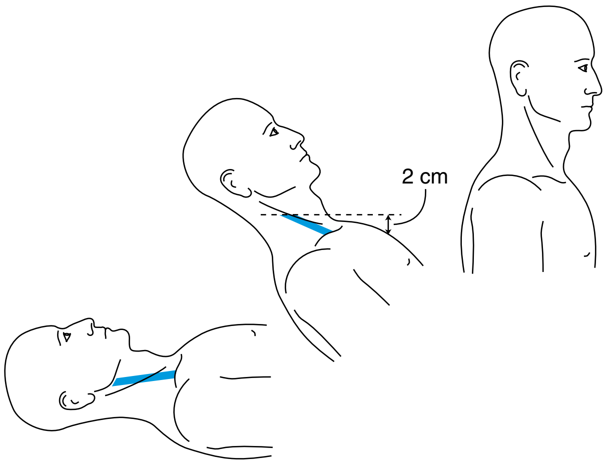


**FIG. 36.1 MEASUREMENT OF VENOUS PRESSURE.** The clinician should vary the patient's position until the top of the neck veins become visible. In this patient, who has normal central venous pressure (CVP), the neck veins are fully distended when supine and completely collapsed when upright. Therefore a semiupright position is used to estimate pressure. In this position the top of the neck veins is 2 cm above the sternal angle, and according to the method of Lewis, the patient's CVP is 2 + 5 = 7 cm water.

most common landmark used in intensive care units and cardiac catheterization laboratories, was originally proposed in the 1940s, when studies showed that using it as the zero point minimized variation in venous pressure of normal persons as they changed position between 0 and 90 degrees.[11]

### C. RELATIVE MERITS OF STERNAL ANGLE AND PHLEBOSTATIC AXIS

Obviously, the measurement of venous pressure is only as good as the reference point used. The phlebostatic axis locates a point in the right atrium several centimeters posterior to the point identified by the method of Lewis (i.e., the zero point using the phlebostatic axis is 9 to 10 cm posterior to the sternal angle; that using the method of Lewis is 5 cm below the sternal angle).[16,17] This means that clinicians using the phlebostatic axis will estimate the CVP to be several centimeters water higher than those using the method of Lewis, even if these clinicians completely agree on the location of the neck veins.

The sternal angle is a better reference point for bedside examination, simply because clinicians can reproducibly locate it more easily than the phlebostatic axis. Even using standard patient positions and flexible right-angle triangles or laser levels, experienced observers trying to locate a point similar to the phlebostatic axis disagreed by several centimeters in both horizontal and vertical directions.[18,19]

## B. ELEVATED VENOUS PRESSURE

### 1. TECHNIQUE

To measure the patient's venous pressure, the clinician should examine the veins on the right side of the patient's neck because these veins have a direct route to the heart. Veins in the left side of the neck reach the heart by crossing the mediastinum,

where the normal aorta may compress them, causing left jugular venous pressure to be sometimes elevated even when CVP and right venous pressure are normal.[20,21]

The patient should be positioned at whichever angle between the supine and upright position best reveals the top of the neck veins (see Fig. 36.1). The top of the neck veins is indicated by the point above which the subcutaneous conduit of the external jugular vein disappears or above which the pulsating waveforms of the internal jugular vein become imperceptible.

### 2. EXTERNAL VERSUS INTERNAL JUGULAR VEINS

Either the external or internal jugular veins may be used to estimate pressure because measurements in both are similar.[22] Traditionally clinicians have been taught to use only the internal jugular vein because the external jugular vein contains valves which purportedly interfere with the development of a hydrostatic column necessary to measure pressure. This teaching is erroneous for two reasons: (1) The internal jugular vein also contains valves, a fact known to anatomists for centuries.[23-25] These valves are essential during cardiopulmonary resuscitation, preventing blood from flowing backward during chest compression,[26] and (2) Valves in the jugular veins do not interfere with pressure measurements because flow is normally toward the heart. Indeed valves probably act like a transducer membranes (e.g., the diaphragm of a speaker), which amplify right atrial pressure pulsations and make the venous waveforms easier to see.[23]

### 3. DEFINITION OF ELEVATED CVP

After locating the top of the external or internal jugular veins, the clinician should measure the vertical distance between the top of the veins and one of the external reference points discussed previously (see Fig. 36.1). The venous pressure is abnormally elevated if (1) the top of the neck veins are more than 3 cm above the sternal angle, (2) the CVP exceeds 8 cm water using the method of Lewis (i.e., >3 cm above the sternal angle + 5 cm), or (3) the CVP is greater than 12 cm water using the phlebostatic axis.

## C. BEDSIDE ESTIMATES OF VENOUS PRESSURE VERSUS CATHETER MEASUREMENTS

### 1. DIAGNOSTIC ACCURACY*

In studies using a standardized reference point, bedside estimates of CVP are within 4 cm water of catheter measurements 85% of the time.[22,30,31] According to these studies, the finding of an elevated CVP (i.e., top of neck veins >3 cm water above sternal angle or >8 cm water using method of Lewis) greatly increases the probability that catheter measurements are elevated (likelihood ratio [LR] = 8.9, EBM Box 36.1). The finding of a normal CVP on examination (<8 cm using the method of Lewis) decreases significantly the probability of a measured CVP greater than 12 cm water (LR = 0.2; see EBM Box 36.1). If disease is defined instead as measured CVP greater than 8 cm, the finding of normal venous pressure on examination is slightly less compelling (LR = 0.3), indicating that some patients with normal venous pressure on examination have modestly elevated measured values (between 8 and 12 cm water†).

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*Studies that test the diagnostic accuracy of bedside estimates of CVP are difficult to summarize because they often fail to standardize which external reference point was used.[27-29]

†For purposes of comparison, "measured pressure" here is in centimeters of water using the method of Lewis. Most catheterization laboratories measure pressure in mm Hg using the phlebostatic axis as the reference point.

**EBM BOX 36.1**
*Inspection of the Neck Veins**

| Finding (Reference)† | Sensitivity (%) | Specificity (%) | Likelihood Ratio‡ if Finding Is Present | Likelihood Ratio‡ if Finding Is Absent |
|---|---|---|---|---|
| ***Estimated Venous Pressure Elevated*** | | | | |
| Detecting measured CVP >8 cm water[22,30-33] | 47-92 | 83-96 | **8.9** | **0.3** |
| Detecting measured CVP >12 cm water[22,30,31] | 78-95 | 67-93 | **6.6** | **0.2** |
| Detecting elevated left heart diastolic pressures[34-36] | 10-58 | 96-97 | **3.9** | NS |
| Detecting low LV ejection fraction[37-39] | 7-25 | 96-98 | **6.3** | NS |
| Detecting MI (if chest pain)[40] | 10 | 96 | 2.4 | NS |
| Predicting postoperative pulmonary edema[41,42] | 19 | 98 | **11.3** | NS |
| Predicting postoperative MI or cardiac death[41,42] | 17 | 98 | **9.4** | NS |
| ***Estimated Venous Pressure Low*** | | | | |
| Detecting measured CVP ≤5 cm water[33] | 90 | 89 | **8.4** | **0.1** |
| ***Positive Abdominojugular Test*** | | | | |
| Detecting elevated left heart diastolic pressures[34,43,44] | 55-84 | 83-98 | **8.0** | **0.3** |
| ***Early Systolic Outward Movement (CV Wave)*** | | | | |
| Detecting moderate-to-severe tricuspid regurgitation[45] | 37 | 97 | **10.9** | 0.7 |

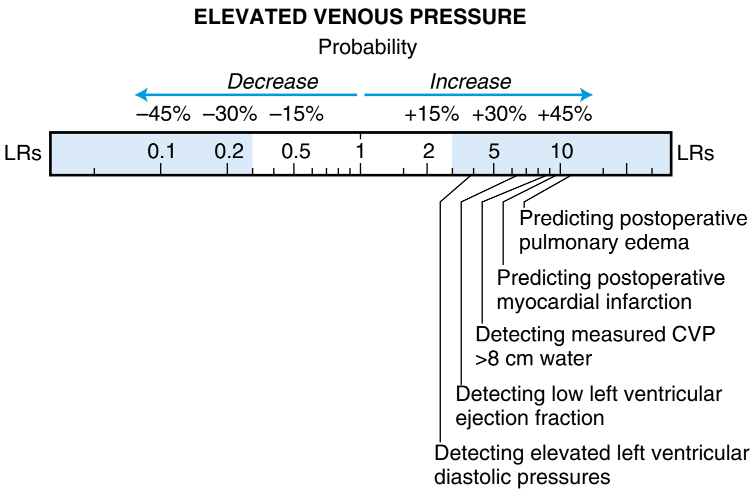
*Diagnostic standard: for *measured CVP*, measurement by catheter in supine patient using method of Lewis[22,30-33] or unknown[31]; for *elevated left heart diastolic pressures* or *low ejection fracture*, see Chapter 48; for *myocardial infarction*, see Chapter 49.

†Definition of findings: for *elevated venous pressure*, bedside estimate >8 cm water using method of Lewis,[22,30,31] >12 cm water using phlebostatic axis,[41,42] or unknown method[34-37]; for *low venous pressure*, estimate CVP ≤5 cm water using method of Lewis[33]; and for *positive abdominojugular test*, see the text.

‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

*CVP*, Central venous pressure; *LV*, left ventricular; *MI*, myocardial infarction; *NS*, not significant

Click here to access calculator




This tendency to slightly underestimate the measured values, which is elucidated further in the following section, explains why estimates made during expiration are slightly more accurate than those made during inspiration: During expiration, the neck veins move upward in the neck, increasing the bedside estimate and minimizing the error.[22]

## 2. WHY CLINICIANS UNDERESTIMATE MEASURED VALUES

Of the many reasons why clinicians tend to underestimate measured values of CVP, the most important one is that the vertical distance between the sternal angle and physiologic zero point varies as the patient shifts position (Fig. 36.2).[5,46] Catheter measurements of venous pressure are always made while the patient is lying supine, whether the venous pressure is high or low. However, bedside estimates of venous pressure must be made in the semiupright or upright positions if the venous pressure is high, because only these positions reveal the top of distended neck veins. Fig. 36.2 shows that the semiupright position increases the vertical distance between the right atrium and sternal angle approximately 3 cm, compared with the supine position, which effectively lowers the bedside estimate by the same amount. The significance of this is that patients with mildly elevated CVP by catheter measurements (i.e., 8 to 12 cm), whose neck veins are interpretable only in more upright positions, may have bedside estimates that are normal (i.e., <8 cm water).

In support of this, even catheter measurements using the sternal angle as reference point are approximately 3 cm lower when the patient is in the semiupright position than when the patient is supine.[47-49]

# D. CLINICAL SIGNIFICANCE OF ELEVATED VENOUS PRESSURE

## 1. DIFFERENTIAL DIAGNOSIS OF ASCITES AND EDEMA

In patients with ascites and edema, an elevated venous pressure implies that the heart or pulmonary circulation is the problem; a normal venous pressure indicates another diagnosis is the cause.

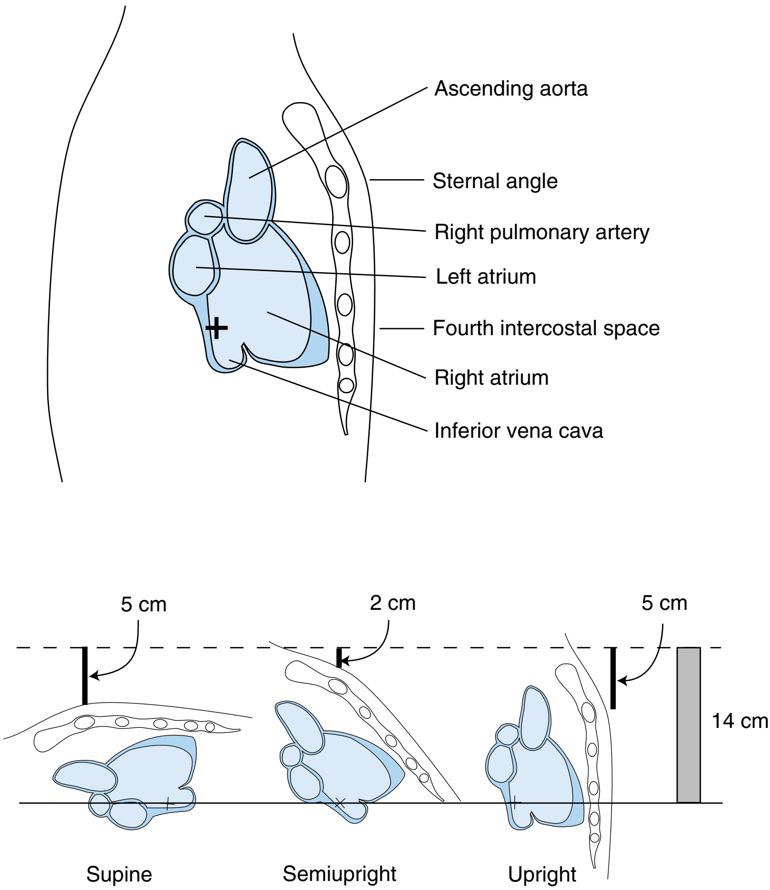



**FIG. 36.2** **CENTRAL VENOUS PRESSURE AND POSITION OF PATIENT.** The top half of the figure shows the sagittal section of a 43-year-old man, just to the right of the midsternal line, demonstrating the relationship between the sternal angle, right atrium, and phlebostatic axis (indicated by the *black cross* in the posterior right atrium). The bottom half of the figure illustrates the changing vertical distance between the phlebostatic axis (*solid horizontal line*) and sternal angle in the supine (0 degrees), semiupright (45 degrees), and upright (90 degrees) positions. The venous pressure is the same in each position (14 cm above the phlebostatic axis, *gray bar on right*) but the vertical distance between the sternal angle and the top of the neck veins changes in the different positions: the vertical distance is 5 cm in the supine and upright positions but only 2 cm in the semiupright position. Using the method of Lewis (see text), therefore the estimate of venous pressure from the semiupright position (7 cm = 2 + 5) is 3 cm *lower* than estimates from the supine or upright positions (10 cm = 5 + 5 cm). Based upon reference 5.

### 2. ELEVATED VENOUS PRESSURE AND LEFT HEART DISEASE

EBM Box 36.1 shows that in patients with symptoms of angina or dyspnea the finding of elevated venous pressure increases the probability of elevated left atrial pressure (LR = 3.9; see EBM Box 36.1)‡ and depressed ejection fraction (LR = 6.3). The opposite finding (normal neck veins) provides no diagnostic information about left heart pressure or function (negative LRs not significant; see EBM Box 36.1). In patients presenting to emergency departments with sustained chest pain, the finding of elevated venous pressure increases the probability of myocardial infarction (MI) (LR = 2.4).

### 3. ELEVATED VENOUS PRESSURE DURING PREOPERATIVE CONSULTATION

The finding of elevated venous pressure during preoperative consultation predicts that the patient—without diuresis or other treatment—will develop postoperative pulmonary edema (LR = 11.3; see EBM Box 36.1) or MI (LR = 9.4).

### 4. ELEVATED VENOUS PRESSURE AND PERICARDIAL DISEASE

Elevated venous pressure is a cardinal finding of cardiac tamponade (100% of cases) and constrictive pericarditis (95% of cases). Therefore the absence of elevated neck veins is a conclusive argument against these diagnoses. In every patient with elevated neck veins the clinician should search for other findings of tamponade (i.e., pulsus paradoxus; prominent *x′* descent but absent *y* descent in venous waveforms) and constrictive pericariditis (pericardial knock, prominent *x′* and *y* descents in venous waveforms) (see Chapter 47).

### 5. UNILATERAL ELEVATION OF VENOUS PRESSURE

Distention of the left jugular veins with normal right jugular veins sometimes occurs because of kinking of the left innominate vein by a tortuous aorta.[20,21] In these patients the elevation often disappears after a deep inspiration.

Persistent unilateral elevation of the neck veins usually indicates local obstruction by a mediastinal lesion, such as aortic aneurysm or intrathoracic goiter.[52]

## E. CLINICAL SIGNIFICANCE OF LOW ESTIMATED VENOUS PRESSURE

Few studies have addressed whether clinicians can accurately detect *low* venous pressure, a potentially difficult issue because *normal* venous pressure is often defined as less than 8 cm water (i.e., *low* and *normal* measurements overlap). Nonetheless, in one study of 38 patients in the intensive care unit (about half receiving mechanical ventilation), the clinician's estimate of a CVP of 5 cm water or less accurately detected a measured value of 5 cm water or less (positive LR = 8.4), an important finding if the clinician is contemplating whether or not fluid challenge is indicated.

# III. ABDOMINOJUGULAR TEST

## A. THE FINDING

During the abdominojugular test, the clinician observes the neck veins while pressing firmly over the patient's mid abdomen for 10 seconds, a maneuver that probably

‡ During cardiac catheterization, a measured right atrial pressure greater than or equal to 10 mm Hg detects a measured pulmonary capillary wedge pressures of greater than or equal to 22 mm Hg with an LR of 3.5, similar to bedside examination (LR = 3.9).[50,51]

increases venous return by displacing splanchnic venous blood toward the heart.[44] The CVP of normal persons usually remains unchanged during this maneuver or rises for a beat or two before returning to normal or below normal[30,43,44,53,54] If the CVP rises more than 4 cm water and remains elevated for the entire 10 seconds, the abdominojugular test is positive.[34,44] Most clinicians recognize the positive response by observing the neck veins at the moment the abdominal pressure is released, regarding a *fall* more than 4 cm as positive.

The earliest version of the abdominojugular test was the **hepatojugular reflux**, introduced by Pasteur in 1885 as a pathognomonic sign of tricuspid regurgitation.[55] In 1898 Rondot discovered that patients with normal tricuspid valves could develop the sign, and by 1925 clinicians realized that pressure anywhere over the abdomen, not just over the liver, would elicit the sign.[53] Several investigators have contributed to the current definition of the abdominojugular test.[30,44,56]

### B. CLINICAL SIGNIFICANCE

In patients presenting for cardiac catheterization (presumably because of chest pain or dyspnea), a positive abdominojugular test is an accurate sign of elevated left atrial pressure (i.e., ≥15 mm Hg, LR = 8; see EBM Box 36.1). Therefore a positive abdominojugular test is an important finding in patients with dyspnea, indicating that at least some of the dyspnea is due to disease in the left side of the heart. A negative abdominojugular test decreases the probability of left atrial hypertension (LR = 0.3; Table 36.1).

## IV. KUSSMAUL SIGN

Kussmaul sign is the paradoxical elevation of CVP during inspiration. In healthy persons venous pressure falls during inspiration because pressures in the right heart decrease as intrathoracic pressures fall. Kussmaul sign is classically associated with constrictive pericarditis, but it occurs in only the minority of patients with

**TABLE 36.1 Distinguishing Internal Jugular Waveforms from Carotid Pulses[57-60]**

| Characteristic | Internal Jugular Vein | Carotid Artery |
|---|---|---|
| Character of movement | Descending movement most prominent | Ascending movement most prominent |
| Number of pulsations per ventricular systole | Two, usually | One |
| Palpability of pulsations | Not palpable or only slight undulation | Easily palpable |
| Change with respiration | During inspiration, pulsations become more prominent but drop lower in neck | No change |
| Change with position | Pulsations lower in neck as patient sits up | No change |
| Change with abdominal pressure | Pulsations may temporarily become more prominent and move higher in neck | No change |
| Change with pressure applied to the neck just below pulsations | Pulsations become less prominent | No change |

constriction [61,62] and is found in other disorders, such as severe heart failure,[62,63] pulmonary embolus,[64] and right ventricular infarction.[65-68]

An excellent video of Kussmaul sign is available.[69]

### A. PATHOGENESIS OF ELEVATED VENOUS PRESSURE, ABDOMINOJUGULAR TEST, AND KUSSMAUL SIGN

The peripheral veins of normal persons are distensible vessels that contain approximately two-thirds of the total blood volume and can accept or donate blood with relatively little change in pressure. In contrast, the peripheral veins of patients with heart failure are abnormally constricted from tissue edema and intense sympathetic stimulation, a change that reduces extremity blood volume and increases central blood volume. Because constricted veins are less compliant, the added central blood volume causes CVP to be abnormally increased.[5]

In addition to causing an elevated CVP, venoconstriction probably also contributes to the positive abdominojugular test and Kussmaul sign, two signs that often occur together. Most patients with constrictive pericarditis and Kussmaul sign also have a markedly positive abdominojugular test; many patients with severe heart failure and a markedly positive abdominojugular test also have Kussmaul sign.[62] The venous pressure of these patients, unlike that of healthy persons, is very susceptible to changes in venous return. Maneuvers that increase venous return—exercise, leg elevation, or abdominal pressure—increase the venous pressure of patients with the abdominojugular test and Kussmaul sign, but not that of healthy persons.[5] Kussmaul sign may be nothing more than an inspiratory abdominojugular test, the downward movement of the diaphragm compressing the abdomen and increasing venous return.[70]

Even so, an abnormal right ventricle probably also contributes to Kussmaul sign because all of the disorders associated with Kussmaul sign are characterized by a right ventricle that is unable to accommodate more blood during inspiration (i.e., in constrictive pericarditis the normal ventricle is constrained by the diseased pericardium, and in severe heart failure, acute cor pulmonale, or right ventricular infarction, the dilated right ventricle is constrained by the normal pericardium). A right side of the heart thus constrained only exaggerates inspiratory increments of CVP, making Kussmaul sign more prominent.[5]

### B. CLINICAL SIGNIFICANT OF KUSSMAUL SIGN

In addition to serving as an important clue to the diagnoses of constrictive pericarditis and right ventricular infarction, Kussmaul sign is associated with an adverse prognosis when found in patients with severe heart failure (LR = 3.5 for 1-year mortality).[71]

## VI. VENOUS WAVEFORMS

### A. IDENTIFYING THE INTERNAL JUGULAR VEIN

Venous waveforms are usually only conspicuous in the internal jugular vein, which lies under the sternocleidomastoid muscle and therefore becomes evident by causing pulsating movements of the soft tissues of the neck (i.e., it does not resemble a subcutaneous vein). Because the carotid artery also pulsates in the neck, the clinician must learn to distinguish the carotid artery from internal jugular vein, using the principles outlined in Table 36.1.

Of the distinguishing features listed in Table 36.1, the most conspicuous one is the character of the movement. Venous pulsations have a prominent *inward* or *descending* movement, the outward one being slower and more diffuse. In contrast, arterial pulsations have a prominent *ascending* or *outward* movement, the inward one being slow and diffuse.

## B. COMPONENTS OF VENOUS WAVEFORMS

Although venous pressure tracings reveal three positive and negative waves (Fig. 36.3), the clinician at the bedside usually sees only two descents, a more prominent *x′* descent and a less prominent *y* descent (Fig. 36.4). Fig. 36.3 discusses the physiology of these waveforms.

## C. TIMING THE *X′* AND *Y* DESCENTS

The best way to identify the individual venous waveforms is to time their *descents*, by simultaneously listening to the heart tones or palpating the carotid pulsation (see Fig. 36.4).

### 1. USING HEART TONES

The *x′* descent ends just *before* $S_2$, as if it were a collapsing hill that slides into $S_2$ lying at the bottom. In contrast, the *y* descent begins just *after* $S_2$.

### 2. USING THE CAROTID ARTERY

The *x′* descent is a systolic movement that coincides with the tap from the carotid pulsation. The *y* descent is a diastolic movement beginning after the carotid tap, with a delay approximately equivalent to the interval between the patient's $S_1$ and $S_2$.[59,75]

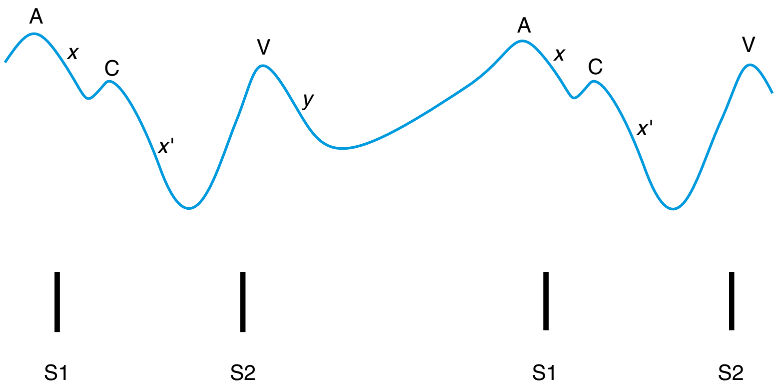



**FIG. 36.3 VENOUS WAVEFORMS ON PRESSURE TRACINGS.** There are three positive waves (A, C, and V) and three negative waves (*x*, *x′*, and *y* descents). The A wave represents right atrial contraction; the *x* descent, right atrial relaxation. The C wave—named "C" because Mackenzie originally thought it was a carotid artifact—probably instead represents right ventricular contraction and closure of the tricuspid valve, which then bulges upward toward the neck veins.[72,73] The *x′* descent occurs because the floor of the right atrium (i.e., the A-V valve ring) moves downward, pulling away from the jugular veins, while the right ventricle contracts (physiologists call this movement the "descent of the base").[74] The V wave represents right atrial filling, which eventually overcomes the descent of the base and causes venous pressure to rise (most atrial filling normally occurs during ventricular systole, not diastole). The *y* descent begins the moment the tricuspid valve opens at the beginning of diastole, causing the atrium to empty into the ventricle and venous pressure to abruptly fall.

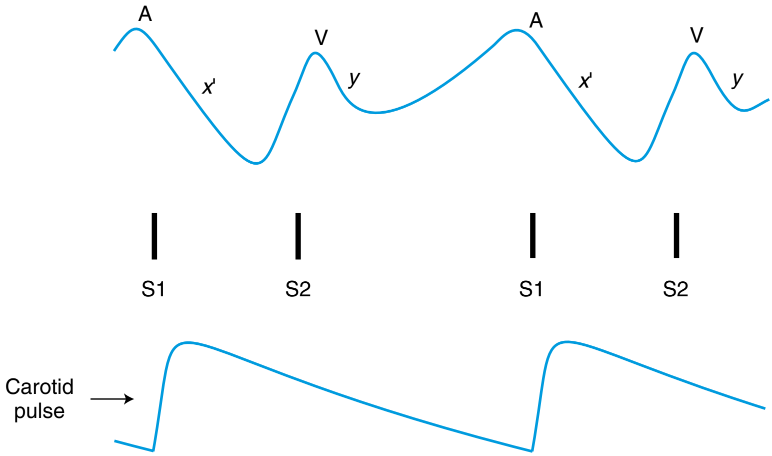



**FIG. 36.4** **VENOUS WAVEFORM: WHAT THE CLINICIAN SEES.** Although tracings of venous waveforms display three positive and three negative waves (see Fig. 36.3), the C wave is too small to see. Instead, the clinician sees two descents per cardiac cycle: the first represents merging of the *x* and *x′* descents and is usually referred to as the *x′* descent (i.e., "*x*-prime" descent). The second is the *y* descent, which is smaller than the *x′* descent in normal persons. The clinician identifies the descents by timing them with the heart tones or carotid pulsation (see text).

## D. CLINICAL SIGNIFICANCE

The normal venous waveform has a prominent *x′* descent and a small or absent *y* descent; there are no abrupt outward movements.[75]

Abnormalities of the venous waveforms become conspicuous at the bedside for one of two reasons: (1) the descents are abnormal, or (2) there is a sudden outward movement in the neck veins.

### 1. ABNORMAL DESCENTS

There are three abnormal patterns: (1) The W or M pattern ($x' = y$ pattern). The *y* descent becomes unusually prominent, which, along with the normal *x′* descent, creates two prominent descents per systole and traces a W or M pattern in the soft tissues of the neck; (2) The diminished *x′* descent pattern ($x' < y$ pattern). The *x′* descent diminishes or disappears, making the *y* descent most prominent. This is the most common abnormal pattern, occurring both in atrial fibrillation (loss of A wave) and many different cardiomyopathies (more sluggish descent of the base), and (3) The absent *y* descent pattern. This pattern is relevant only in patients with elevated venous pressure because healthy persons with normal CVP also have a diminutive *y* descent.

The etiologies of each of these patterns are presented in Table 36.2.

### 2. ABNORMALLY PROMINENT OUTWARD WAVES

If the clinician detects an abnormally abrupt and conspicuous outward movement in the neck veins, the clinician should determine if the outward movement begins just before $S_1$ (presystolic giant A waves) or after $S_1$ (tricuspid regurgitation and cannon A waves).

**TABLE 36.2 Venous Waveforms**

| Finding | Etiology (Ref.) |
|---|---|
| **ABNORMAL DESCENTS** | |
| W or M pattern ($x' = y$) | Constrictive pericarditis[58,76*] |
| | Atrial septal defect[77-79] |
| Diminished $x'$ descent ($x' < y$) | Atrial fibrillation |
| | Cardiomyopathy[75] |
| | Mild tricuspid regurgitation |
| Absent y descent[†] | Cardiac tamponade[58] |
| | Tricuspid stenosis[80] |
| **ABNORMALLY PROMINENT OUTWARD WAVES** | |
| Giant A wave (presystolic wave) | Pulmonary hypertension[58] |
| | Pulmonic stenosis[58] |
| | Tricuspid stenosis[80,81] |
| Systolic wave | Tricuspid regurgitation[45,82-84] |
| | Cannon A waves[58] |

*The prominent *y* descent of constrictive pericarditis is sometimes called *Friedreich's diastolic collapse of the cervical veins* (after Nikolaus Friedreich, 1825–1882).
†If venous pressure is normal, the absence of a *y* descent is a normal finding; however, if venous pressure is elevated, the absence of the *y* descent is abnormal and suggests impaired early diastolic filling.

## A. GIANT A WAVES (ABRUPT PRESYSTOLIC OUTWARD WAVES)

Giant A waves have two requirements: (1) sinus rhythm and (2) some obstruction to right atrial or ventricular emptying, usually from pulmonary hypertension, pulmonic stenosis, or tricuspid stenosis.[57,58,81] Nonetheless, many patients with severe pulmonary hypertension lack this finding, because their atria contract too feebly or at a time in the cardiac cycle when venous pressures are falling.[79,85]

Some patients with giant A waves have an accompanying abrupt presystolic sound that is heard with the stethoscope over the jugular veins.[86]

## B. SYSTOLIC WAVES

**(1). TRICUSPID REGURGITATION.** In patients with tricuspid regurgitation and pulmonary hypertension, the neck veins are elevated (more than 90% of patients) and consist of a single outward systolic movement that coincides with the carotid pulsation and collapses after $S_2$ (i.e., prominent *y* descent).[82-84] Some patients have an accompanying midsystolic clicking sound over the jugular veins.[87] Because the jugular valves often become incompetent in chronic tricuspid regurgitation, the arm and leg veins also may pulsate with each systolic regurgitant wave (see Chapter 46).

The finding of early systolic outward venous waveforms (CV wave) greatly increases the probability of moderate-to-severe tricuspid regurgitation (LR = 10.9; see EBM Box 36.1).

**(2). CANNON A WAVES.** Cannon A waves represent an atrial contraction that occurs just after ventricular contraction, when the tricuspid valve is closed[§]. Instead of ejecting blood into the right ventricle, the contraction forces blood upward into the jugular veins. Cannon A waves may be regular (i.e., with every arterial pulse) or intermittent.

***(A). REGULAR CANNON A WAVES.*** This finding occurs in many paroxysmal supraventricular tachycardias (fast heart rates) and junctional rhythms (normal heart rates), both of which have retrograde P waves buried within or just after the QRS complex.[58]

***(B). INTERMITTENT CANNON A WAVES.*** If the arterial pulse is regular but cannon A waves are intermittent, only one mechanism is possible: atrioventricular dissociation (see Chapter 16). In patients with ventricular tachycardia the finding of intermittently appearing cannon A waves detects atrioventricular dissociation with a sensitivity of 96%, specificity of 75%, positive LR of 3.8, and negative LR of 0.1 (see Chapter 16).[88]

If the arterial pulse is irregular, intermittent cannon A waves have less importance because they commonly accompany ventricular premature contractions and less commonly atrial premature contractions (see Chapter 16).

*The references for this chapter can be found on* www.expertconsult.com.

[§] The electrocardiographic correlate of the cannon A wave is a P wave (atrial contraction) falling between the QRS and T waves (ventricular systole).

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